IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of	)	
	)	
Schwiebert *et al.*	)	Art Unit: 1616
	)	
Application No. 10/542,555	)	Examiner: John D. Pak
	)	
Filing Date: August 29, 2005	)	Confirmation No. 7032
	)	
For: METHODS AND COMPOSITIONS FOR P2X RECEPTOR CALCIUM ENTRY CHANNELS AND OTHER CALCIUM ENTRY MECHANISMS	)	

DECLARATION UNDER 37 C.F.R. § 1.132

Mail Stop Amendment
Commissioner for Patents
P. O. Box 1450
Alexandria, VA 22313-1450

BALLARD SPAHR ANDREWS & INGERSOLL
Customer No. 23859

Sir:

I, Erik M. Schwiebert, Ph.D., hereby declare that:

1. I received my BA degree in Biology from Grinnell College (1987). I earned my Ph.D. degree in Physiology 1992 from Dartmouth College. After my postdoctoral fellowships at Dartmouth Medical School (1992-1993) and the Johns Hopkins University School of Medicine (1993-1996), I joined the faculty at UAB in December 1996. In October 2002, I was promoted to Associate Professor. I have over 20 years of experience in the field of physiology, with an emphasis on epithelial cell physiology and pathophysiology. Over the past 12 years, I have studied the purinergic ligand, ATP, in the study of cystic fibrosis. More recently (over the past 5 years), I have studied the biometal ligand, zinc, and how it interacts with receptors and channels. This includes specific experience in purinergic receptors. A partial curriculum vitae is attached to this declaration as an exhibit (**Exhibit A appended**).

2. I have reviewed the specification of the above-identified application, and the specification of PCT/2004/01298, filed January 20, 2004; as well as the specification of 60/441,045, filed January 17, 2003, and 60/474,4223, filed June 3, 2003, to which the above-identified application claims priority.

3. I have reviewed the Office Action mailed February 7, 2008 in connection with the above-identified application and the following references cited in that Office Action:

a. Taylor et al.(The Journal of Clinical Investigation, October 1999, Vol. 104 No. 7, pages 875-884, cited as disclosing that P2X purinergic receptor channels bind ATP and
mediate Ca2+ influx and signals that stimulate secretory CI- transport across epithelia.

b. Schwiebert et al. (American Journal of Physiology, June 2001), by *, and *, cited as disclosing that P2X receptors, on binding their ATP ligand, may
increase cytosolic Ca2+ transiently and stimulate CI and fluid secretion and ciliary beat
frequency.

c. CAPLUS abstract 2001:30580, cited as disclosing that the zinc ion is a known P2X receptor modulator that potentiates the actions of ATP in P2X receptor gated ion channels.

d. Boucher, Jr. (US 6,926,911; hereinafter, Boucher), cited as disclosing treating cystic fibrosis patients with meglumine chloride, i.e. N-methyl D-glucamine chloride, via multiple delivery means.

e. Senior (Medline abstract 86146262), cited as disclosing the role of pH in cystic fibrosis.

f. Rubenstein et al. (US 2002/0115619), cited as disclosing that cystic fibrosis is known to be treated with combination therapy wherein beneficial effects of multiple individual agents are combined.

4. I understand that claims 1-3, 12-13, 21-23, and 142-146 have been rejected under 35 U.S.C. § 103(a) as being obvious. Specifically, I understand that the rejection is based in part on the contention that the ordinary skilled artisan in this field would have been motivated to utilize the zinc ion, to treat cystic fibrosis. I present in this declaration evidence indicating that this is not the case, that those of skill in the art at the time of

the invention would not have believed that treatment with Zn^{2+} and ATP; α, β-methylene-ATP; benzoyl-benzoyl-ATP; ATPγS; or AMPPNP, would result in a sustained elevation in cytosolic Ca^{2+} levels in the cell.

5. As an expert in the field of epithelial cell physiology and pathophysiology of the biometal zinc in general and treatment of cystic fibrosis in particular, and as an individual with extensive knowledge of the level of understanding of those of skill in the art at the time Application Serial No. 10/542,555 was filed, I believe that no one would have thought cell calcium levels would be stimulated and kept in a sustained elevated state in the cell via this or any maneuver. The mammalian cell is designed to fight to keep calcium levels low and all calcium agonists elicit transient calcium spikes. Our prior work, cited above as prior art, elicited transient rises in cell calcium with ATP alone. The same is elicited in past publications by zinc alone. The combination of the two ligands, zinc and ATP, in combination with the chemical and pH moficiations to the saline vehicle (no magnesium, no sodium, Mg and Na replaced by NMDG, and a pH of 7.9) was the critical "secret sauce" that elicited marked and sustained increases in cell calcium, not heretofor observed by us or by others with these agonists. Previous to this accidental finding designed to define what P2X receptor channels were specifically being activated by zinc and ATP, we did not feel that our cellular responses were sufficient enough to warrant working toward a CF therapy (e.g., in particular, the brief and transient nature of the cell calcium response). In fact, our Journal of Biological Chemistry paper that was published after our provisional patent was filed was reviewed by Science (only 10% of submitted articles are reviewed) and was well received. The only point of contention that precluded its publication in Science was that the reviewers were astounded in terms of how we could possibly be causing such a large and sustained calcium signal that remains elevated as it does. More recent unpublished work (done before I vacated my academic laboratory) defined other reasons why we believe that we trigger the sustained cell calcium signal in order of the cellular events in time:

(1) Zinc and ATP in the modified saline solution collaborate to gate or open P2X4 and P2X6 heteromultimeric purinergic receptor calcium entry channels, allowing calcium influx from extracellular stores.

(2) Zinc also stimulates one or more zinc-sensing G protein-coupled receptors to trigger calcium release from intracellular or internal ER stores.

(3) Zinc emptying of ER stores causes further store-operated calcium entry through another known cellular pathway for calcium mobilization.

(4) Zinc enters into airway epithelial cells where it may inhibit the plasma membrane calcium ATPase pump that pumps cell calcium out of cells to bring cell calcium levels back to normal low levels.

Having said all that, removal of zinc and ATP causes a full reversal of the effect (i.e., it is not irreversible). If it was not a reversible biological effect on cell calcium, we would have not filed a patent or pursued the work so vigorously as a therapy. Simply put, this is a novel method and composition not heretofor realized.

I declare that all statements made herein of my own knowledge and belief are true and that all statements made on information and belief are believed to be true, and further, that the statements are made with the knowledge that willful false statements are punishable by fine or imprisonment, or both, under section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Date: 8-4-2008

Dr. Erik M. Schwiebert, Ph.D.

EXHIBIT A

Partial CV: Biosketch and Past Research Support for Dr. Erik M. Schwiebert, Ph.D.

(Next 6 Pages)

Principal Investigator/Program Director (Last, First, Middle): SCHWIEBERT, Erik Mills, Ph.D,

BIOGRAPHICAL SKETCH

Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2.
Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

NAME	POSITION TITLE
Erik M. Schwiebert, Ph.D.	Chief Executive Officer, Chief Scientific Officer and Director
eRA COMMONS USER NAME SCHWIEBERTE	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	YEAR(s)	FIELD OF STUDY
Grinnell College	B. A.	1983-1987	Biology
Dartmouth College	Ph.D.	1987-1992	Physiology
Dartmouth College	Post-doc	1992-1993	Physiology
John Hopkins University School of Medicine	Post-doc	1993-1996	Physiology

A. Positions and Honors:

1997-2002 Assistant Professor of Physiology and Biophysics, University of Alabama at Birmingham (UAB)
1997-2002 Assistant Professor of Cell Biology (secondary appointment), UAB
1997-2007 Research Scientist in the Gregory Fleming James CF Research Center
2002-2007 Associate Professor of Physiology and Biophysics, UAB
2002-2007 Associate Professor of Cell Biology (secondary appointment), UAB
2005-2007 Research Scientist in the Nephrology Research and Training Center
2005-2007 Research Scientist in the Recessive Polycystic Kidney Disease Research & Translational Core Centers
2007-Present Chief Executive Officer, Chief Scientific Officer and Director, DiscoveryBioMed, LLC

1993 John W. Strohbehn Medal for Excellence in Biomedical Research from Dartmouth Medical School
1993-1996 NIH NRSA Postdoctoral Fellowship Award (4 Years)
1996 W. Barry Wood Young Investigator Award for Excellence in Biomedical Research from Johns Hopkins University
School of Medicine (Nominated by Peter Agre, M.D.)
1996 Biophysical Society Membranes Subgroup Award
1996 Pew Scholars Nominee for UAB
1997 Symposium Speaker for Experimental Biology 1997
1997-2002 Editorial Board for the American Journal of Physiology Cell Physiology
1999-2002, 2004-Present Editorial Board for the American Journal of Physiology Renal Physiology
2000 Symposium Speaker for Experimental Biology 2000
2001 Ad-Hoc Reviewer for Medical Biochemistry (MEDB) Study Section
2000-2005 Ad-Hoc and Permanent Member of NIDDK Subcommittee B Review Committee for Diabetes, Endocrinology and Metabolism
2002-2004 Speaker and Session Chair for the 2002, 2003 and 2004 CF Foundation Williamsburg Conferences and the 2002, 2003 and 2004 North American CF Conferences
2003 Symposium Speaker for Experimental Biology 2003
Guest Editor for a "Current Topics in Membranes" Volume for Academic Press
2004 and 2005 Ad-Hoc Reviewer for NIDDK Special Emphasis Panel ZDK1 GRB-R O1 on "Short Programs in Multidisciplinary Research Training"
2004 Symposium Speaker for Purines 2004
2004 Named Associate Editor for *Purinergic Signaling*
2005 Chair and Reviewer, NIDDK Special Emphasis Panel ZDK-1 GRB-R (M3) Diabetes and Autoimmunity
2005 Editorial Board, *BioMedCentral-Physiology*
2005 Ad-Hoc Reviewer for Special Emphasis Panel ZAG-1 ZIJ-4 (O3) "Alzheimer's Disease Cellular Models
2005 Reviewer for Special Emphasis Panel ZRG1 RUS-D (O2) "Renal Transport and PKD Sciences."
2005 Reviewer for PPG SEP "Epithelia" for NHLBI
2006 Reviewer for PPG RUS-G(02) "Renal Ion Transport"
2006-Present American Physiological Society (APS) Awards Committee
2006 Session Leader and Symposium Speaker for Purines 2006 in Ferrara, Italy
2006 Reviewer for NIDDK Special Emphasis Panel ZDK1 GRB-B J1 R Short Term Education Programs for Under-represented Persons (STEP-UP R25)
2007 Introductory Speaker and Platform Speaker for EB 2007 Symposia
2006-Present Permanent Member of NIDDK Subcommittee D Review Committee for Kidney, Hematologic and Urologic Diseases
Fall 2007 Guest Editor, Special Issue for Purinergic Signaling entitled "Physiology of Nucleotide Release"

Fall 2007 Class Member of the Entrepreneurial Accelerator Program, Birmingham Venture Club and Bradley, Arant, Rose and White, LLP
Fall 2007 Lecturer, UAB School of Business, Idea to IPO, Genesis of DiscoveryBioMed, LLC
Fall 2007 Lecturer, New York Academy of Sciences, Idea to IPO, Genesis of DiscoveryBioMed, LLC

B. Publications (Selected from 66 Original Papers, 26 Reviews/Book Chapters, 3 Patents, and 9 IPDs)

Light DB, **Schwiebert EM**, Karlson KH, and BA Stanton. Atrial natriuretic peptide inhibits a cation channel in renal inner medullary collecting duct cells. *Science* 243: 383-385, 1989.
Light DB, **Schwiebert EM**, Fejes-Toth G, Naray-Fejes-Toth A, Karlson KH, McCann FV, and BA Stanton. Chloride channels in the apical membrane of cortical collecting duct cells in culture. *Am. J. Physiol.* 258: F273-F280, 1990.
Schwiebert EM, Light DB, Fejes-Toth G, Naray-Fejes-Toth A, and BA Stanton. A GTP-binding protein activates chloride channels in a renal epithelium. *J. Biol. Chem.* 265: 7725-7728, 1990.
Schwiebert EM, Karlson KH, Friedman PA, Dietl P, Spielman WS, and BA Stanton. Adenosine regulates a chloride channel via protein kinase C and a G protein in a rabbit cortical collecting duct cell line. *J. Clin. Invest.* 89: 834-841, 1992.
Schwiebert EM, Gruenert DC, and BA Stanton. GTP-binding proteins inhibit cAMP activation of chloride channels in cystic fibrosis airway epithelial cells. *Proc. Natl. Acad. Sci. USA* 89: 10623-10627, 1992. *(Comm. by Gerhard Giebisch)*
Kunzelmann K, **Schwiebert EM**, Zeitlin PL, Kuo W-L, Stanton BA, and DC Gruenert. An immortalized cystic fibrosis tracheal epithelial cell line homozygous for the delF508 CFTR mutation. *Am. J. Respir. Cell Mol. Biol.* 8: 522-529, 1993.
Schwiebert EM, Mills JW, and BA Stanton. Actin-based cytoskeleton regulates a chloride channel and cell volume in a renal cortical collecting duct cell line. *J. Biol. Chem.* 269: 7081-7089, 1994.
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McCoy DE, **Schwiebert EM**, Karlson KH, Spielman WS, and BA Stanton. Identification and function of A_1 adenosine receptors in normal and cystic fibrosis human airway epithelial cells. *Am. J. Physiol.* 268: C1520-C1527, 1995.
Fulmer SB, **Schwiebert EM**, Morales MM, Guggino WB, and GR Cutting. Two cystic fibrosis transmembrane conductance regulator mutations have different effects on both pulmonary phenotype and regulation of outwardly rectified chloride currents. *Proc. Natl. Acad. Sci. USA* 92: 6832-6836, 1995. *(Comm. by Victor McCusick)*
Hwang TH, **Schwiebert EM**, and WB Guggino. Apical and basolateral ATP stimulate tracheal epithelial chloride secretion via multiple purinergic receptors and signaling pathways. *Am. J. Physiol.* 270: C1611-C1623, 1996.
Hanaoka K, Devuyst O, **Schwiebert EM**, Wilson PD, and WB Guggino. Role of CFTR in autosomal dominant polycystic kidney disease. *Am. J. Physiol. Cell Physiol.* 270: C389-C399, 1996.

Morales MM, Carroll TP, Morita T, **Schwiebert EM**, Devuyst O, Wilson PD, Lopes AG, Stanton BA, Dietz HC, Cutting GR, and WB Guggino. Both wild-type and a functional isoform of CFTR are expressed in kidney. *Am. J. Physiol.* 270: F1038-F1048, 1996.
McNicholas CM, Guggino WB, **Schwiebert EM**, Hebert SC, Giebisch GH, and ME Egan. Sensitivity of a renal K^+ channel (ROMK2) to the inhibitory sulfonylurea compound, glibenclamide, is enhanced by co-expression with the ATP-binding cassette transporter CFTR. *Proc. Natl. Acad. Sci. USA* 93: 8083-8088, 1996. *(Comm. by Robert Berliner)*
Devuyst O, Burrow CR, **Schwiebert EM**, Guggino WB, and PD Wilson. Developmental regulation of CFTR expression during human nephrogenesis. *Am. J. Physiol.* 271: F723-F735, 1996.
Schwiebert EM, Potter ED, Hwang T-H, Ding C, Qiu W, Guggino WB, Levine MA, and SE Guggino. Cyclic nucleotide-gated chloride and sodium currents in rat tracheal airway epithelium. *Am. J. Physiol.* 272 (41): C911-C922, 1997.
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McNicholas CM, Nason MW, Guggino WB, **Schwiebert EM**, Hebert SC, Giebisch GH, and ME Egan. A functional CFTR-NBF1 is required for ROMK2-CFTR interaction. *Am. J. Physiol.* 273: F843-F848, 1997.
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Zsembery A, Boyce AT, Liang L, Peti-Peterdi J, Bell PD, and **EM Schwiebert**. P2X nucleotide receptors are calcium entry channels that trigger a sustained increase in cytosolic calcium in human airway epithelial cells. *J. Biol. Chem* 278(15): 13398-13408, 2003.

Jones JR, **Schwiebert EM**, DuVall MD, Venglarik CJ, Wen H, Kovacs T, Mazur M, Clancy JP, Braunstein GM, Bates E, Greer H, Maddry JA, and EJ Sorscher. Activation of chloride secretion in cystic fibrosis cells and tissues by the substituted imidazole SRI 2931. *Biochemistry* 42: 13241-13249, 2003.

Zsembery A, Bebok Z, Fortenberry JA, Liang L, Tucker TA, Boyce AT, Braunstein GM, Hanson EL, Rice WC, Bell PD, and **EM Schwiebert.** Extracellular zinc and ATP-activated P2X receptor calcium entry channels restore chloride secretion in cystic fibrosis. *J. Biol. Chem.* 279: 10720-10729, 2004.

Braunstein GM, Zsembery A, Tucker TA, Schwiebert LM, and **EM Schwiebert.** Purinergic signaling underlies CFTR modulation of human airway epithelial cell volume. *J. of Cystic Fibrosis* 3(2): 99-117, 2004.

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Hovater MB, Olteanu D, Hanson HL, Cheng NL, Siroky B, Fintha A, Komlosi P, Liu W, Satlin LM, Bell PD, Yoder BK, and **EM Schwiebert**. Loss of apical monocilia on collecting duct principal cells impairs apically-directed ATP release and flow-induced calcium signals in an integrated system. *Purinergic Signal.* 4(2): 155-170, 2008.
Banizs B, Komlosi P, Bevensee MO, **Schwiebert EM**, Bell PD, and BK Yoder. Altered intracellular pH regulation and Na/HCO3 transporter activity in choroid plexus of the cilia defective Tg737orpk mutant mouse. *Am. J. Physiol.* 292(4): C1409-C1416, 2007.
Richards-Williams C, Contreras JL, Berecek KH, and **EM Schwiebert**. Extracellular ATP and zinc activate ATP-gated and zinc-modulated P2X purinergic receptor channels expressed by rodent primary islets and β-cell models to induce insulin secretion. *Purinergic Signal.* In Press, 2008.
Schwiebert EM, Lopes AG, and WB Guggino. Chloride channels along the nephron. *Current Topics in Membranes: Chloride Channels*; ed. WB Guggino, Volume 42, Chapter 10, pp 265-315, Academic Press, Inc., San Diego, CA, c1994.
Schwiebert EM and WB Guggino. Abnormal chloride and sodium channel function in cystic fibrosis airway epithelia. Chapter 195, The Lung: Scientific Foundations; Eds. RG Crystal, JB West, et al. Lippincott-Raven Publishers, Philadelphia c1996.
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Schwiebert EM, Benos DJ, and CM Fuller. Cystic fibrosis: A multiple exocrinopathy caused by dysfunctions in a multifunctional transport protein. *Am. J. Med.* 104 (6): 576-590, 1998.
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Schwiebert EM and BK Kishore. "Extracellular purinergic signaling along the renal epithelium." *Am. J. Physiol. (Renal, Fluid, Electrolyte Physiol.)* 280: F945-F963, 2001.
Schwiebert EM and A Zsembery. "Extracellular ATP as a signaling molecule for epithelial cells." *Biochim. Biophys. Acta* 1615: 7-32, 2003.
Schwiebert EM, Liang L, Cheng NL, Williams CR, Olteanu D, Welty EA, and A Zsembery. Extracellular zinc and ATP-gated P2X purinergic receptor channels: New zinc receptors as physiological sensors and therapeutic targets. Purinergic Signal. 1(4): 299-310, 2005.
Hovater MB, Olteanu D, Welty EA, and **EM Schwiebert.** Purinergic signaling in the lumen of the normal nephron and in remodeled PKD encapsulated cysts. *Purinergic Signal* 4(2): 109-124, 2008.
Schwiebert EM and JG Fitz. Purinergic signaling microenvironments: An Introduction. *Purinergic Signal.* 4(2): 89-92, 2008.

C. Research Support

EM Schwiebert resigned his tenured faculty appointment as an Associate Professor of Physiology and Biophysics and of Cell Biology at the University of Alabama at Birmingham, effective 9-30-2007. Effective 10-1-2007, an active R01 award, 1 R01 DK67343-03, was transferred from Dr. Schwiebert to a collaborator on this R01 award, Dr. Mark O. Bevensee, Ph.D. Likewise, a Juvenile Diabetes Research Foundation (JDRF) award was transferred to Dr. Dale J. Benos, Ph.D. for the remainder of this 1-year award.

Dr. Schwiebert has no active research support from the NIH or from any private foundation.
Please see Other Support for a complete listing of past funding.

OTHER SUPPORT
Erik M. Schwiebert, Ph.D.

Active

Dr. Schwiebert has no active NIH or private foundation awards or grants.

Pending

12-1-2008 through 5-31-2009
1 R43 DK 083171-01 Phase 1 SBIR PI: Erik M. Schwiebert, Ph.D.
Organization: DiscoveryBioMed, Inc.
"Sodium Transport Inhibitors for Hypertension and Cystic Fibrosis"
Total Costs: $100,000

This R43 application received a 136 priority score for CMBK study section.

Past Research Support over the Last 3 Years

NIH NIDDK 1 R01 DK 067343-04A2 (PI: E. Schwiebert)

"Ion Transport Dysregulation in Cilium-deficient ARPKD"
9-1-2005 through 8-31-2010
Direct Costs: $184,500 for Year 4 Total Direct Costs: $943,000
4.2 PM
Working Hypothesis: Loss of apical central monocilium-derived signals in ductal epithelia causes upregulated ENaC- and NHE-mediated Na^+ absorption and resultant hypertension.

Overlap: None
Note: Transferred in 03 of the award to Dr. Mark O. Bevensee, Ph.D., a central collaborator on the award.

Juvenile Diabetes Research Foundation Innovation Grant 5-2007-262 (PI: E. Schwiebert)
"Zinc and ATP as Cytoprotective Autocrine Ligands for Beta Cells and Islets"
2-1-2007 through 1-31-2008
Direct Costs: $100,000 (1 Year Only) Total Direct Costs: $110,000
1.8 PM
Working Hypothesis: Extracellular ATP and zinc, co-secreted with insulin in response to the glucose stimulus, augment insulin secretion and are cytoprotective for beta cell and islet health and function.

Overlap: None
Note: Transferred the award to Dr. Dale J. Benos, Ph.D. as mandated by Dr. Benos, Chair of Dr. Schwiebert's former Department. Award is completed.

NIH NIDDK P30 DK072482-01A1 (PD: E. Sorscher)
"UAB CF Research and Translation Core Center"
1.8 PM

5-1-2007 through 4-30-2012
Direct Costs: $129,130 for Cell Model and Assay Core A – K. Kirk PI) Total Direct Costs: $501,265
E. Schwiebert was an "investigator" slated to help with this Core on behalf of the P30 and the CF Center.

Overlap: There is overlap involving CFTR electrophysiology with the P50 below that will end shortly.
Note: Role abandoned for this P30 PPG.

NIH NIDDK P30 DK 074038-02 (PD: L. Guay-Woodford)

"UAB Recessive Polycystic Kidney Disease Translational Core Centers"
9-1-2005 through 8-31-2010
Direct Costs: $80,000 (for Physiology Core C) Total Direct Costs: $400,000
0.55 PM
E. Schwiebert was co-Director of the Cellular Physiology Resource (Core C of this P30 PPG) and a participant in the Administrative Core.

Overlap: None
Note: Role abandoned for this P30 PPG.

NIH NIDDK 1 R01 DK 060065-04 (PI: J. Collawn)

"Cell Biology of CFTR in Polarized Epithelia"
12-1-2002 through 11-30-2007
Direct Costs: $250,000 for Current Year Total Direct Costs: $1,250,000
0.24 PM
This project investigates recycling of CFTR at the cell surface.

Overlap: None.
Note: This grant is in competitive renewal.

NIH NIDDK P50 DK 062397-10 (PD: E. Sorscher)

"SCOR Mechanistic Studies into CF Pathogenesis and Chloride Secretion"
9-1-2002 through 8-31-2007
Direct Costs: $30,040 (for Assay Core B) Total Direct Costs: $151,700
1.8 PM
This grant investigates basic mechanisms of CF pathogenesis and intervention.

Overlap: This P50 grant ended 08/31/2007 and will not be renewed.

Mentor for Fellowship Grants:

NIH NIGMS MARC Program NRSA 1 F31 GM078758-01 (PI: C. Williams, Ph.D. Candidate, Mentor: E. Schwiebert)
"Zinc and ATP: Autocrine Signalling Molecules for Beta Cells"
9-1-2006 through 8-31-2009
Direct costs: $29,382 Total Direct $88,416
0.0 PM

Overlap: None

Note: Dr. Schwiebert remains the sponsor and mentor; Dr. Kathleen Berecek, Ph.D. is the co-sponsor and co-mentor along with Dr. Juan Contreras, M.D.